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NEPHRITIS OF THE NEWLY BORN.

An Address delivered before the Medical Society of the District of Columbia, Nov. 28, 1895.

BY

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NEW YORK.

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NOTHING would have pleased me more than to appear before you, who have kindly consented to listen to me part of an evening, with something absolutely new. The history of medicine, however, exhibits but very few instances of striking novelty. It is more replete with the proofs of a slow and steady evolution than with sudden and unthought-of revelations. Still, there is one peculiar feature both in the study of our science and the practice of our art—viz., that wherever we approach it it is intensely interesting. That is why even the men borne down with hard work, and altogether too often near the brink of mental and physical exhaustion in the performance of their arduous daily duties, are always roused to enthusiasm by a single new experience, an unheard-of fact, a novel hypothesis, or only a new point of view calculated either to enlarge their horizon or to benefit their fellow men.

To me the connection of the kidneys with the rest of the organism has been a subject of interest through all my professional life. These organs are so intimately interwoven with the whole physiological existence that either their anatomy or their function participates in every disease of every organ. This is particularly perceptible in the infectious diseases, no matter whether mild or severe. In many of them one of the forms of nephritis is very common. In scarlatina, for instance, the desquamative process is quite active in the uriniferous tubes, and results in a peculiar form of inflammation; in some cases of scarlatina and most of the other acute eruptive and infectious maladies it is parenchymatous changes that are more frequently met with. Thus, indeed, it is worth while to study the urine in every case of disease. It is true that we are not always rewarded with the finding of severe lesions; for, happily, most of the cases of secondary nephritis are neither dangerous nor of long duration. But there is none of them but may lead to a severe form, with possibly a fatal termination. Therefore the frequency of infectious diseases in infancy and childhood ought to fix our attention constantly in the direction of the kidneys. It is true that sometimes we are unable to find anything but albuminuria, which, in the absence of kidney elements under the microscope, we are liable to dismiss as transient and of little account. But in this we are very apt to be mistaken. My cases of uncomplicated and transient albuminuria have become wonderfully scarce since I invariably employ for the examination of the urine the centrifuge. Among twenty successive cases where the verdict is "trace of albumin" I am certain to find in the centrifuged deposits of nineteen, within a few minutes, the almost uniform result—blood-cells, hyaline casts, hyaline casts studded with epithelia, or finely or coarsely granulated casts.

Many of these forms of nephritis are, as I said, short-lived. Quite often will they disappear within a week or ten days. But this happy termination is far from being universal. There is nobody here but has been surprised in a child of advanced age or in an adolescent by an attack of uræmic convulsion, the cause of which could be traced to a scarlet fever which, six or ten years ago, terminated in apparent recovery. The same experience is had with nephritis from other causes; for, unfortunately, we know by this time that besides scarlatina, measles, varioloid, and varicella, even vaccinia, acute local diseases of the skin, erysipelas, rheumatism, typhoid fever, acute and chronic intestinal diseases may be complicated with or followed by nephritis. For this reason nephritis is very common in infancy and childhood, and ought to be searched for whenever the origin of prominent or dangerous symptoms is not at once clear. Fortunately, it is easy to obtain a specimen of urine, for catheterization is more readily successful in the child than in many adults. Thus it will frequently happen that a nephritis is found when the prominent cerebral symptoms suggested the diagnosis of encephalitis or meningitis. Of the many cases of this nature which I have met with, the following will furnish an illustration:

A boy of five weeks who had appeared to be in fair health was taken with high fever and convulsions. The case occurred in a family living in very moderate circumstances, therefore the medical man had good reason to suppose that the infant had been ailing some days before it was considered necessary to call him in. The temperature was 104° to 105° F., the pulse almost uncountable, and the convulsions had not been frequent when I saw the patient. There was some cyanosis and perspiration over the upper part of the body; the legs and feet were cold, the head was very hot. There was no œdema. The pupils were equal, fairly dilated, contracted a very little, but sluggishly, under the influence of

a strong ray of light, and under the same light dilated again and contracted within certain limits. The equality of the pupils, combined with that peculiar floating condition of the iris, made me think of uræmia as the cause of all the cerebral symptoms. The urine was known to be scanty, but that is what it also is in meningitis, and in every child that has not been supplied with a sufficient quantity of water. Fortunately, there was some in the bladder. Boiling almost solidified it, and the microscope revealed blood-cells, epithelial and granular casts, the latter both fine and coarse. The child died; no autopsy could be had. No clew could be found to the causation of the fatal disease; and still, the baby was so young that in all probability the origin of the fatal nephritis might have been found in some occurrence of the first few days of life.

It is this period of early life to which I mean to direct your special attention to-night by reporting a few of the many cases of nephritis met with within a few days or weeks after birth. Some are primarily renal diseases, some are secondary. To the latter class belong those nephritides which are complicated with or dependent upon *intestinal disorders.* This connection is quite frequent. In many instances diarrhœal disorders are the *results* of nephritis, but quite frequently both acute and chronic intestinal diseases appear to be the *causes* of nephritis, which may be quite ominous; for indeed it is here as in other diseases, many of which are liable to terminate fatally by their renal complication. Every practitioner loses many a case of pneumonia, not through the severity of the pulmonary lesion, but on account of the accompanying nephritis. In this way the entero-colitis of the newly born is quite apt to destroy life through nephritis. In a highly creditable essay (*Arch. f. Kinderk.*, 1894, xvii, p. 222) Felsenthal and Bernhard have studied the connection of nephritis with acute and chronic intestinal disorders of infancy and child-

hood. They have also collected the literature on the subject. Parrot met with it in the atrophy ("athrepsia") of young infants; Kjellberg, Fischl, Stiller, Baginsky, Hirschsprung, Hagenbach, Henoch, Epstein, and others have recorded cases of nephritis accompanying intestinal disorders. The cases of this description are by no means rare in the first week of life. When I look over the list of the numerous cases of the kind I have personally seen, it almost seems to me supererogation to record a case; and still I know that many of my colleagues with whom I saw the cases appeared to be surprised at recognizing both the presence of nephritis in such cases and the facility with which the diagnosis could be made.

The literature on the subject is but scanty. I have, however, reason to believe that even those who have known the connection between intestinal diseases and nephritis quite well have not published their experience. It has happened to me personally that my chapters on catarrh and ulceration of the bowels in my *Intestinal Diseases of Infancy and Childhood*, 1887, are silent on that subject by an oversight of my own. But in the discussion on Two Cases of Acute Primary Nephritis in Infancy, by L. Emmett Holt, one of which was perhaps caused by intestinal sepsis without that explanation being suspected, I took occasion to say (*Trans. of the Am. Pæd. Soc.*, 1891, vol. iii, p. 233): "There are cases of nephritis which complicate intestinal diseases. It is true that many spells of vomiting and diarrhœa are merely symptoms of nephritis. A number of cases supposed to be cholera, even Asiatic, are found to be acute nephritis. On the other hand, where we have to do with an acute or subacute intestinal catarrh, a *prolonged seizure may give rise to secondary nephritis.* I am positive that it will be found to be much more frequent than it was considered to be." In the *Archives of Pædiatrics*, June, 1890, p. 420, diarrhœa

is also briefly mentioned by me as one of the many causes of nephritis.

It is but two years ago that a colleague presented a boy, five days old, the child of very poor parents, at my office. The cord had fallen off and the stump looked normal. The mouth was slightly covered with sprue. The lips, fingers, and toes were cyanotic, though the feeble heart appeared normal; the baby was nearly collapsed. Rectal temperature, 103° F. For two days there had been loose mucous discharges in great numbers; they were slightly offensive, did not contain meconium any more, but already at that early time coagulated masses of casein. There was no tenesmus and no blood. The urine of the second and third day appeared to the attendants darker than normal; during the last day but little had been passed. We drew about ten cubic centimetres of a dark, smoke-colored fluid. It contained albumin in great quantity, and under the microscope blood-cells, epithelial and granular casts, and urates. The baby died the following day. No autopsy.

It was a similar case that I saw with the same gentleman a few months afterward. He made the diagnosis before I met him. It proved one of the most fortunate I have seen; firstly, because it was not so severe as the former, and, secondly, because there was ample time to restore and equalize by warm bathing both the cutaneous and general circulation, to cleanse and disinfect the intestine and fill the blood-vessels, to establish a flow of urine through the uriniferous tubes by means of copious and frequent irrigations of the bowels, and to stimulate the heart by judicious doses of strychnine, of which the infant took nearly a milligramme during twenty-four hours.

What little I have said of the nature of the discharges, their offensiveness and frequency, suggests the cause of the secondary nephritis. It evidently depends on the absorption of a toxine, no matter whether it originates in the

invasion of a streptococcus, or of the bacterium coli, or one of the other forms of microbes detailed by Booker and by Jeffries in the *Transactions of the American Pædiatric Society* of 1889.

Their absorption is facilitated by some peculiar anatomical conditions.

The muscular apparatus of the intestine of the fœtus and of the newly born is but slightly developed. During fœtal life its function is but trifling, and its contents move but slowly. Immediately after birth that muscular debility predisposes to colic, as air which is swallowed, and gases, both innocuous and putrid, which are developed in the tract, are expelled with difficulty. Besides, the infantile digestive tract is unexpectedly long. According to Beneke, the proportion of the length of the body to that of the small intestine is in the adult 100 to 450; in the newly born, however, 100 to 570; in the second year, 100 to 660. Moreover, the villi are generally numerous and large; some assert they surpass in size those found in the adult intestine; the capillaries of the villi, it is claimed, have greater absolute size, so much so that their diameter is larger than that of the same vessels in the adult.* All this tends to show that both the accumulation of septic material in, and absorption from, the interior of the intestines is rendered very easy. The access of microbes to the intestinal tract of the newly born is by no means difficult. How they enter, through the mouth, the anus, or the blood, I have but recently discussed in the first number of *Pædiatrics*. After all, it seems that the nephritis originating from intestinal infection is of a similar nature to what we observe in typhoid fever or any of the other infectious diseases.

* A. Jacobi. *Intest. Dis. of Infancy and Childhood.* George S. Davis, 1887. Chapter on Intestinal Digestion.

Nephritis in *typhoid fever* of the newly born I have seen but once, for the simple reason that I have observed but this one case of typhoid fever in one so young. It was cursorily mentioned on page 29 of my *Treatise on Diphtheria*, 1880. The baby died on the sixteenth day of its life, twenty-two years ago. The mother recovered. Its kidneys were much congested, the two substances hardly discernible from each other, and blood oozed from the cut surfaces. There had been anuria for two days, and no urine was found in the bladder after death.

In one of the three cases of *diphtheria* in the newly born, reported on page 30 of my *Treatise*, I was favored with an autopsy. The baby was taken seven days after birth and died on the ninth. The kidneys were in the condition described in the previous case. No microscopical examination of the urine could be had.

In connection with this subject I now present the case of the youngest patient I have seen destroyed by *potassic chlorate:*

B. C., a boy of nine days, was seized, January 15, 1882, with convulsions, after not having voided urine for several hours. The last time, when a teaspoonful was passed, it was of a dark color, stained the napkin, and seemed to give pain during the discharge. There was constant rectal tenesmus, with some protrusion of the bowel, some five or six hours before the convulsion. During all this time the complexion was sallow, and the lips and finger and toe nails were blue. I saw the infant after the convulsions, with hardly a pulse, bluish lips, brownish complexion, the scleræ still yellow and largely ingested with dilated blood-vessels. Heart beats from 200 to 220 a minute, scarcely perceptible. Within an hour after my visit he died. The blood in the whole body was of an intensely dark color, the heart of normal size and structure, ductus Botalli nearly closed, ductus venosus Arantii still open. Lungs and spleen were engorged and purplish, so

was the liver. The kidneys were large; a number of blood points—small hæmorrhages—were visible on the longitudinal section; there were, besides, a number of dark streaks corresponding with the uriniferous tubes, and the difference between the two renal substances was almost extinct. Their color was unusually dark, and they offered a strongly marked elastic resistance to the touch. What little urine (about two cubic centimetres) was taken from the bladder contained much pelvic epithelium, and consisted almost exclusively of decomposed blood-cells.

The great resemblance of this form of nephritis to what I had described in the third volume of Gerhardt's *Handbuch der Kinderkrankheiten*, article Diphtheria, in 1877, and in a paper on The Remedial and Poisonous Effects of Chlorate of Potassium, published in the *Medical Record* of March 15, 1879, made me inquire rather scrupulously into the history of the dead baby. The mother had suffered from copious vaginal discharge during the last few months of her pregnancy. Neither she nor her surroundings were of the cleanest. The first few days of the infant's life were normal. On the third and fourth day sprue developed and covered lips and cheeks with thick deposits. The midwife in charge called no physician. She knew the best thing for sprue and inflicted it. She brushed the mouth with a saturated solution of potassic chlorate, as she proudly asserted, quite often, and frequently gave a few drops to drink. I could not learn the strength of her solution. She always used it and it had a powerful effect, she said. As far as I was permitted to learn, she dissolved a tablespoonful in a tumblerful of water; I still found a sediment of the salt in the bottom of a tumbler.

A case of nephritis after *vaccination* was reported by Perl in the *Berliner klinische Wochenschrift*, 1893, No. 28. It behaved exactly as nephritis in infectious fevers. The child, two years and nine months old, became very restless about the usual time of the onset of a vaccinia fever—viz., from the fourth to the fifth day; at the same time there seemed to be abdominal and lumbar pains.

Within a day after, simultaneously with the appearance of six vaccination vesicles, there was albumin in the urine to the amount of one half of a per mille; also hæmatin, blood-cells, and some leucocytes. The casts were either purely hyaline, or hyaline studded with epithelium. The child was well on the twelfth day. The whole morbid process ran its full course in six days, with no serious symptoms at all.

The following is a case of a similar description in a very young infant:

In an immigrant hotel of Greenwich Street, New York, I saw with Dr. John Bishop, April 4, 1877, two children, one of four years and one of three weeks, who had been vaccinated ten days previously. I was expected to see the older one, who had an erysipelas of moderate size and severity; it got well after twice traveling over the surface of the body. On the very day of my visit the baby, who had run through her vaccinia fever with no unusual discomfort, was seized with an attack of convulsions. When I saw her there was a rectal temperature of 103°, a dazed look, injected conjunctivæ, pupils equal, somewhat dilated, and floating under the influence of light. The latter symptoms induced me to draw urine and examine it. It was scanty and contained a trace of albumin, a few blood-cells, and hyaline and finely granular casts. This nephritis lasted two weeks before it finally disappeared. During all this time there was no other convulsion, no œdema, but an occasional vomiting spell and diarrhœa during the first week of the illness; the pupil symptom persisted; the temperature varied between 101° and 103°, a moderate remission taking place in the morning. During the second (and last) week of the disease all the above symptoms gradually disappeared, and the temperature went down. In their place a slight œdema of the lower extremities and of the face were observed. The microscopical changes in the condition of the urine remained the same about ten days after they were first discovered. Then they disappeared, and recovery remained undisturbed.

Renal disorders, more or less dangerous, are direct results of *sudden changes in the circulation*, without or with visible alterations of the blood. To the first class belongs a case I once saw with a medical friend who had so much confidence in the vitality and vigor of the newly born that he commenced to enforce his theories on the necessity of early hardening immediately after birth. He would plunge the newcomers into cold water, and feel a grim delight in taking their incipient breath away and making them shriek in reflex self-defense. Two of his victims I saw with him; they died within a fortnight. The second we examined post mortem. There was a pneumonia, it is true, perhaps sufficient to destroy life. But the most apparent and probable cause of death, preceded by suppression of urine, was evidently bilateral nephritis. Both the kidneys were large, intensely congested, and blood poured out of the cuts; the difference between the two substances could not be distinguished. With him I saw no more such cases, for I suggested the probability that the cold bathing of the newly born furnished us the specimen. But the more I have seen of similar cases in the adult, the more do I feel that I was correct in my charge. For acute nephritis, interstitial, sometimes hæmorrhagic, is an occasionally unavoidable occurrence in sudden suppression of cutaneous circulation. Who has not seen death occurring from nephritis, not preceded by a chronic affection, in persons who have been resuscitated from drowning in an ice cold river, or have been exposed to a driving rain storm while exerting themselves to get under shelter, or to cold and sleet in an open sleigh? What the slow influence of cold can not accomplish in the healthy and vigorous, what not even a nephrectomy can accomplish in the remaining kidney, its sudden effect on the feeble, or fatigued, or even the vigorous, will easily bring about. No matter whether the reasons are to be sought

for in an antagonism of the skin and kidneys, or the enforced elimination of cutaneous excrements through the kidneys, the facts are actual. Moreover, direct experiments made by Lassar unmistakably prove the causation of interstitial inflammation by sudden refrigeration.

Like excessive *cold*, *heat* may lead to nephritis and death. Only once have I seen a newly born sacrificed in that way through his first bath. The midwife evidently had anæsthesia or analgesia. Bystanders noticed the steaming of the water in the bath tub, the suffering of the suffocating baby, his livid appearance; and the raising of large blisters on the surface told the story. The baby died within a day, having lost some blood mixed with meconium and passed no urine. Even the bladder was empty at the autopsy, and deeply congested. The kidneys were livid and succulent; blood oozed out of the cut surfaces. Blood was also extravasated under the capsules. If the case had run a longer course, in all probability hæmoglobinuria, produced by dissolution of blood-corpuscles, would have shown itself, as in the experimental researches of Ponfick and of Wertheim. Changes in the general circulation need not, however, be of this sudden and violent type, and still result in some injury.

Indeed, the albuminuria of the newly born is frequently due to the *insufficiency of circulation*, and passes off when the latter is freely established; just as the venous obstruction caused by heart or lung disease results in temporary albuminuria in the adult. In a certain number of these cases of almost congenital albuminuria there is no blood under the microscope, in others there is, in others there is more—viz., nephritis. It is probable that after most cases of protracted *asphyxia* of the newly born albumin will be found in the urine, with or without blood. Thus the kidneys repeat but the process which has been so much bet-

ter studied in the brain by Langdon Down* and also by me.†

Indeed, in three cases of nephritis, two of which proved fatal, observed within five weeks after birth, no ætiology except that of previous long continued asphyxia could be elicited. It was in those two that granular and coarse casts were in the majority; in the one which survived, there was still after weeks blood and a few epithelial and finely granular casts.

In *congenital heart diseases* with cyanosis, albuminuria is quite common. Again I warn against the facility of overlooking it. Time and again I am told there is no albumin in a specimen; time and again there is in such cases a trace, which is called "only a trace," but yields fields full of different casts in the centrifuged specimen. This very trace is sometimes not discovered unless the test tube be looked at through water, and unless some little time is given for the coagulation to become visible. Nephritis does not always work with heavy loads of albumin; that the last stage of chronic nephritis of any period of life may be without albuminuria for weeks in succession need not be retold.

I once saw a baby of four months, who had *spina bifida* and consecutive paralysis and contractures of both lower extremities, die with nephritis. We seldom see our patients with spina bifida when they breathe their last; for, until a brief time ago, most of them were left to die without an attempt at relieving them, and a neighboring medical man was called in at the last minute so that a certificate of death might be obtained. The same opportunity of observing a fatal case of nephritis in a little girl of three months I had about the same time. The patient had a *paralysis* of both

* *Transactions of the Obstetrical Society*, London, 1876.

† *American Journal of Obstetrics*, xxiv, 1891, No. 6.

lower extremities, dating from birth, and occasioned, probably, by an intraspinal hæmorrhage caused during difficult extraction in breech presentation. Maybe I am correct when in both cases I attribute the renal changes, chronic in character, to the fact that the circulation being impeded by the muscular inactivity of a large part of the body was more directed toward the internal organs. Maybe, however, this suggestion does not appear acceptable,* for it is possible to assume that the same violence which caused a spinal hæmorrhage and paraplegia was sufficient to produce the same effect in the kidneys.

In the newly born we observe not only the adverse results of the sudden changes from fœtal to post-natal circulation, but also lesions depending upon the *peculiar structure of the blood-vessels.* The newly born is removed from the embryo and fœtus by a single station only. Its tissues are in part still embryonic, and endowed with less solid structure. This is why hæmorrhages are so very frequent in the newly born. Meningeal hæmorrhages are most frequent during the first week, and the slight coagulability of the blood of the newly born adds to its dangers. In regard to the brain, I have considered this question years ago, and frequently since, mostly in connection with asphyxia in the newly born. A large number of cases of idiocy, epilepsy, paralysis, and insanity in the very young are due to meningeal hæmorrhage of early days often ushered in by asphyxia. Similar occurrences take place in other organs. Disseminated pleural and pericardial hæmorrhages are quite frequent in the newly born under the influence of retarded or interrupted circulation. When the latter improves, the hæmorrhagic points may become absorbed. So it is in the kidneys.

* As above stated, not even the removal of a whole kidney results in a nephritis of the other.

Parenchymatous hæmorrhages are capable of causing inflammations in the kidneys as they do in other organs. In many cases, however, they prove innocuous. In the muscles, the brain, the lungs, extravasations take place without leaving any trace behind. It is probable that whenever no healthy tissue is torn, when an extravasation takes place between fibrillæ, absorption takes place. When there is, however, an actual lesion of tissue, a secondary inflammation is or may be the consequence. Many years ago I was startled by an acute nephritis appearing in a delicate but healthy boy of four years, the son of a well-known practitioner in New York. None of the usual causes of the disease could be traced, and I was perfectly at sea until a crop of petechiæ appeared over the chest and the extremities. I then learned that six months previously the child had had another attack of purpura which gave rise to no symptoms, and that a few days before the first symptoms of this acute renal disorder there had been a few petechiæ all over the surface. The urine showed under the microscope rather an unusual amount of blood, together with plenty of blood casts and granular casts. It struck me, therefore, that the nephritis was in this case due to disseminated renal hæmorrhages, and I ventured to give a rather favorable prognosis. It took but a few weeks before the patient had fully recovered. Two similar cases have been encountered since, one in a girl of seven, one of eleven years. Both recovered. Never before did it occur to me to look upon the kidneys as more than very rare participants in a purpuric process, except in cases of actual hæmaturia.

In two newly born infants I have seen similar processes originating from the same source. A boy of five days was seen for *melæna* on his fifth day. There was vomiting of blood; there were bloody stools. Their color was not quite black; some of the blood was red, and its origin could

be assigned to the lower part of the intestinal tract. The baby appeared to recover a little from the sudden shock of the loss of blood, when, on the next day, slight traces of blood appeared in the urine. Part of the blood cells were tolerably normal. Within another day the quantity of urine diminished greatly and assumed a smoky hue. The microscope still revealed blood cells, but also blood casts, a very few epithelial and many more finely granular casts. The baby died and the kidneys were removed. Both of them were markedly congested. On the walls of the pelvis were superficial hæmorrhages; sections revealed a number of rather fresh blood points. There was no doubt in the minds of all those present that the nephritis in this case was due to the irritation set up by the local hæmorrhages.

Another case dates twenty-six years back. After a protracted labor a boy was born in breech presentation. Ecchymoses over the abdomen proved the difficulty of parturition and the summary procedures of the midwife in charge. Almost the first urine voided by the infant was bloody, and the diagnosis of traumatic renal hæmorrhage appeared justified. Within a day the blood disappeared almost entirely, and urine became suppressed. The baby died on the fourth day, and was subjected to a coroner's inquest. There was a moderate amount of blood clot under the peritoneal covering of the liver, the liver was torn to a distance of about three centimetres, the peritonæum slightly torn, and blood had escaped into the abdominal cavity. Both kidneys were large, dark, and blood-stained on section; the two substances hardly differed from each other.

These were extreme cases, and their diagnosis was in a short time followed by death. How many there may occur in which extravasation is but moderate, and the amount of local or perhaps unilateral nephritis is not immediately fatal, perhaps even inclined to get well, is difficult to say.

Large maternities, however, and foundling institutions are better prepared for observing such occurrences than the practitioner engaged in private or consulting work.

Frequent causes of nephritis of the newly born are *uric-acid infarctions.* They occur from the second to the twenty-third day, but also before birth.* They are of different varieties. In a part or in all of the straight uriniferous tubes there are found yellowish-red or brownish, spherical or angular bodies in such quantities as to form considerable deposits and, when they are discharged during life, to cause large stains of more or less solidity in the napkins. They are in rare cases accompanied with blood. They consist of uric acid and of ammonium urate. The latter is readily soluble in acetic acid, from which uric acid crystallizes in rhombic shapes. In one case Ebstein met in the tubuli contorti with yellow globules consisting of uric acid and an organic stroma which contained no mucus, but consisted of albuminoids which were soluble in acetic acid, and exhibited either a concentric structure or irregular layers. At once the question rises in our minds as to the nature of this organic stroma. It must strike us that it can be of either of two origins. It is either depending on a

* Virchow's original opinion, according to which the presence of uric-acid infarction requires a certain duration of life, has been to a certain extent rescinded by the proof furnished by a premature and stillborn fœtus which contained uric acid in its urine and urate of ammonium as sediment. Moreover, well-developed uric-acid infarctions were observed by Martin (*Jenaische Ann.*, 1850) in a fœtus born in the unruptured membranes after an unsuccessful attempt at respiration. Hoogeweg (Casper, *Viertelj.*, 1855) met with them in an infant whose heart ceased to beat three quarters of an hour before delivery. Birch-Hirschfeld has a similar case, and Hofmann (*Gerichtl. Med.*, fifth edition, 1891, p. 748) published the cases of two infants, one of whom lived but twenty-three hours, the other only fifteen minutes, who exhibited uric-acid infarctions in full development.

cause not connected with the presence of the uric-acid infarction, or it is the direct consequence of a local irritation caused by the deposit—viz., secondary exudation. In this manner that form of infarction would, by itself alone, exhibit a mild degree of nephritis.

A second form of renal infarctions is of a *hæmorrhagic and pigmentous* nature. They look very much like those already described, and are found in the same localities. They are granular, spherical, or irregular conglomerates, which contain crystals of hæmatoidin. They are the results of small extravasations originating in general hyperæmia of the canaliculi, and depend on various causes, to the principal of which I shall return. The usual changes of hæmatin alter the color of these deposits, which contain no crystals of uric acid or ammonium urate, and are not affected by acetic acid.*

Calcareous deposits are also found in the newly born. They occur mainly in the lower end of the straight canaliculi, near the papillæ, are of a whitish color, and may therefore be mistaken on inspection for interstitial indurations. They are mostly either carbonate or phosphate of calcium, but rarely triple phosphate, and are soluble in

* Crystals of hæmatoidin (=bilirubin) were found by Virchow as early as 1847 (*Verhandl. d. Ges. f. Geburtsh. in Berlin*, vol. ii) in the kidneys, the tissues, and the blood of infants who died while suffering from icterus neonatorum. Their main location is in the renal epithelium and in the uriniferous tubes, but rarely in the urine. They are also found in the fibrinous coagula of the heart, in the parenchyma of the liver (Orth), and in the adipose tissue of the omentum (Neumann). Even in macerated fœtuses they were met with by Neumann and Ruge. It appears, therefore, that at the time of birth, and soon after, ilirubin exists in the blood and tissues (with or without jaundice) in a sufficient quantity to permit its getting free in crystalline form even after death. The presence of genuine uric-acid infarctions is not influenced by his phenomenon and they and bilirubin may occur simultaneously or separately.

dilute hydrochloric acid. They are, under favorable circumstances, deposited into and upon the epithelia.

Which are these favorable circumstances? Both phosphates and carbonates of calcium are known to be deposited from the blood whenever circulation is retarded or impeded; for instance, in the older baby in the latter stages of epiphyseal rhachitis. In the newly born the circulation is retarded or impeded by congenital (or rapidly acquired) heart disease, by general debility, or by asphyxia. As early as 1883 (*Virch. Arch.*) Litten counted among such favorable conditions a coagulation necrosis occasioned by the interruption of circulation. Thus these forms of retarded circulation, to which I alluded before in a different connection, exert a baneful influence from a chemical point of view.

The normal frequency of uric acid and other renal infarctions explains the great many cases of gravel and stone in the very young. They are observed in the earliest age, contrary to the opinion of Rosenstein. This great author on the diseases of the kidneys repudiates the connection between the symptoms of renal colic and vesical calculi, and between renal infarctions and vesical calculi. He admits having observed renal colic in the first year of life, but in a single baby only. Now this is very unfortunate, and can be explained only, I believe, by some characteristics in the field of his observations. Exceptional cases, such as those of Woehler and Denis, in which a renal calculus consisting of uric acid was found in a premature and stillborn fœtus, need not be counted at all. But the observations of Heusinger relating to the frequent occurrence of renal calculus in the first year of life are more conclusive. I met with renal calculus quite frequently when I had more opportunities to make autopsies of young infants, and have often alluded to a series of forty post-mortem examinations

made on babies who died of miscellaneous diseases, in six of whom I found a renal calculus. Nor do I believe I am mistaken when I express my conviction that many of you have observed actual gravel in the very young, and many more the violent spasmodic pains of infants, accompanied with erections, dysuria, even convulsions, and sudden relief mostly attended with urination.

It is evident that the presence of crystalline masses in the tubes and papillæ of the kidneys is liable to be dangerous. They encroach upon the soft tissue in which they are imbedded, disintegrate the epithelium, irritate the surface, and produce slight hæmorrhage and inflammation. In many cases of nephritis of the very young there was a distinct history of dysuria and of copious deposits in the napkins, not infrequently mixed with blood. What gravel and stone can accomplish in more advanced months and years is more easily brought about in the half-perfected tissue of the newly born.

In regard to the dangers attending the presence of uric acid in the kidneys I have more to say on preventives than curatives. When we deal with gravel and stone in the kidneys of adults our efforts are directed to the solution of the deposits. Plenty of water, alkaline mineral waters, alkalies, mainly potassic salts, lithia, piperazine, and lysidine are pressed into service. In the newly born, in whom we must, as infarctions are the rule, expect the presence of the danger, we are in the habit of doing absolutely nothing, though prevention be within easy reach. Water is, if not the panacea, at all events the indicated remedy. But in no period of life is water more withheld from the helpless creature than in the first few days. Mother's milk is not forthcoming until a few days have passed by, and then it appears in small quantities only. Even the experience that the newly born loses weight by being starved is charged

against Providence, which has willed it so from times ante diluvial. If water were given plentifully and as methodically as syrup of figs or castor oil, much harm could be avoided. And here permit me a few words *pro domo*. In regard to feeding the newly born, I have practised these forty years, and taught thirty-five, not only that the very young infant must be fed, but that its artificial food must be greatly diluted. In those early times I knew only that the baby would best bear great dilutions, and I mixed a part of boiled milk with four or five parts of water, or rather of a thin cereal decoction. The latter have at last been recognized as correct, even by Heubner, whose main labors for years have been spent on studying and discussing the question of artificial infant food. But he still sets his face against what he calls "Jacobi's exorbitant dilutions." In the light of what I have had the honor of saying to-night, I profess to have even in those remote times taught better than I knew. At those times I considered the question of digestion only when I recommended large dilutions. It is only a dozen years ago, perhaps, that I began to consider the question of high dilution of the food of the newly born from the point of view of its beneficence in renal infarction and its consequences. In 1887 I spoke of its indications for the purpose of dissolving and eliminating uric-acid infarctions in my *Intestinal Diseases of Infancy and Childhood*. I can assure, as I said then, that since my advice of greatly diluting the food of the newly born, and giving plenty of water from the beginning, has commenced to be minded, I am sadly deprived of the many cases of gravel, dysuria, shrieking spells, and consecutive nephritis which were so common in former times.

The connection of *icterus* of the newly born with local changes in the kidneys is of vital interest. In the adult this intimate dependency upon each other is rare, though

many gross anatomical changes are equally found in all ages. To that class belong septic infection, syphilis of the liver, cirrhosis of the liver of whatever origin, obliterations of the biliary ducts, thrombosis of the portal vein, and catarrh of the duodenum and choledochus duct.

In the newly born many undoubted cases of icterus are due to the destruction of red blood-cells in the first few days, and to the transformation of hæmatin into hæmatoidin (identical with bilirubin). Some of the latter comes from the many ecchymoses and stagnations, both in the skin and the subcutaneous tissue, due to the process of parturition.

The destruction of blood-cells in the newly born is a normal occurrence. According to Hayem and Hélot the blood-cells of the newly born are subject to rapid disintegration. According to Hofmeier the normal congregation of the blood-cells is absent; they exhibit a greater resistance to solving liquids; the number of leucocytes is very changeable, and the size of the blood-corpuscles is very variable. Silbermann found many blood-cells pale, others of normal color in their periphery only; many of various sizes—macro- and microcytes. He also met with nucleated blood-cells in the liver, the spleen, and the bone marrow; with cells of the liver, sometimes also of the spleen, and of the bone marrow containing blood; with red bodies of the club and biscuit form, evidently changed blood-cells; and finally an increase of leucocytes. All of these observations appear to prove the destructibility of the blood of the newly born, which is only equaled, or perhaps even surpassed, by the effect of chronic poisoning, in part observed for experimental purposes.*

* Toluylendiamine, according to Afanassiew and Stadelman, exhibits the following results: Dissolution of red blood-corpuscles and consecutive hæmoglobinuria; increase of the coloring matter of the bile; anæmia; moderate fatty degeneration of the large glands; acute parenchymatous nephritis; destruction of renal epithelia. At the same time

By many the jaundice of the newly born is attributed to absorption of bile into the blood directly from the biliary ducts into the small vessels of hepatic circulation. By others a congenital narrowness of the choledochus duct or an accumulation of mucus in the biliary ducts, or œdema of the periportal connective tissue, or venous obstruction in the liver and consecutive compression of biliary ducts were claimed as the causes of jaundice. Quincke explained it by the patency of the ductus venosus Arantii, and by absorption of bile from the meconium of the intestines.

Meconium is rich in bilirubin. The latter is stored in it during and after the third month of intra-uterine life. Biliverdin accompanies it to such a large amount that Simon (*Arch. f. Gynäk.*, 1875) met with four per cent. of it.

This bilirubin and biliverdin are very liable to be absorbed through the open ductus venosus Arantii, which remains patent in seventy-seven per cent. of all the newly born until after the first week of their lives. Its circulation is free, its blood liquid, and there is a direct communication from the intestinal circulation with that of the vena cava.*

the epithelia of the spleen and liver are seriously damaged either directly by the (experimental) poison or by the circulation of an altered blood. The urine contains copious conglomerate crystals, which probably are not organic, but consist of calcium sulphate.

* Some communication of the same kind, with the same effect, is brought about between the hæmorrhoidal plexus of the rectum (through the hæmorrhoidal vein) and the vena cava, thus circumventing the liver. Still, it must be remembered that less absorption takes place in the rectum than in the rest of the intestinal tract. Absorption is very much more active in the upper part of the large intestine. Kühne knew, 1868 (*Physiol. Chem.*), that icterus may originate in absorption from the colon; and in the small intestines both the amount of meconium and the absorbability of its bilirubin and biliverdin are much greater.

Through the open ductus venosus Arantii the coloring matter of the bile enters the circulation of the whole body, circumventing the liver to such an extent that in some cases of icteric newborn infants it does not participate in the jaundice at all, and produces different degrees of icterus. When peristalsis is active, circulation and absorption are so in proportion, and icterus is early; when peristalsis is but sluggish, and meconium retained unusually long, icterus may appear at a late period. In premature babies the ductus venosus is large, and jaundice liable to be early and very intense. When Elsässer, however, found it closed in three cases no jaundice was observed. Immediately after birth the coloring matter of the bile is considerably increased, and therefrom results another additional cause of jaundice. Besides, as it has been stated, there is no period of life in which under normal circumstances so many blood-cells undergo rapid disintegration. Therein lies another cause for the formation of bilirubin, and for a direct thrombotic interruption of circulation in the smallest blood vessels. Finally, there is no period of life when elimination is less active than during the first days of life. At that time the urine is very scanty, the water supply mostly neglected, and the accumulation of effete material the rule.

Moreover, bilirubin is but scantily dissolved in the fluids of the tissues of the newly born; even in strongly alkaline solutions it is but slightly soluble, according to Hoppe-Seiler. Thus it is that the coloring matter of bile is met with in the urine of the newly born in the shape of the yellow masses (*masses jaunes*) which have already been mentioned in connection with urinary infarctions.

When the absorbed and deposited masses are but scanty they may be eliminated without any symptoms. When there is enough of them to result in a local irrita-

tion, they will cause albuminuria, which is often found in ill-nourished icteric babies. When there is enough to cause thromboses, which are quite common in the capillaries of the portal system, and obstruction of circulation, they give rise to hæmorrhages or to inflammations. As far as the kidneys are concerned, there is a peculiar anatomical reason why nephritis is very liable to appear in the very young.

The post-fœtal growth of blood-vessels and tissues varies considerably. It is least in the common carotid, largest in the renal and femoral arteries. The renal artery and the kidneys, however, do not develop proportionately; the transverse section of the former increases out of proportion to the volume and weight of the latter. Thus it seems that this disproportion between the size of the artery and the condition of the renal tissue establishes a predisposition to congestive and inflammatory conditions of the organ. Moreover, the resistance in the capillary net of the young kidney is unusually great. Experiments prove that the permeability of the capillaries is greater, and that within a given time a proportionately larger amount of water can be squeezed through them in the adult than in the young. This anatomical difference seems, therefore, to be an additional reason why renal diseases are so much more frequent in infancy and childhood, from all causes, with the only exception of that which is reserved for the very last decades of natural life—viz., atheromatous degeneration.*

In conclusion, Mr. President, permit me to recapitulate in a few words the main points of this paper:

Nephritis is a frequent disease of infancy and childhood and by no means very rare in the newly born. What

* Heart and Blood-vessels in the Young. By A. Jacobi, M. D., *Brooklyn Med. Jour.*, March, 1888.

was formerly considered mere albuminuria, or a transient form of it, we have been taught by improved methods of investigation, mainly by the use of the centrifuge, to recognize as nephritis. A predisposition to nephritis in the young is caused by the fragility of the blood vessels in the newly born; by the relative imperviousness of the young renal capillaries compared with the large size of the renal arteries; by the feebleness of the young intestinal muscle, which proves insufficient to expel toxic contents; by the extensiveness and size of the young intestinal blood-vessels and lymphatics and the large size of the villi, all of which favor the absorption of toxines.

From an ætiological point of view, nephritis in the newly born may be:

1. *Congestive* (from feeble circulation, congenital heart disease, asphyxia, or exposure to low temperatures).

2. *Obstructive* (from the physiological rapid decomposition of the blood of the newly born; the formation of hæmatoidin=bilirubin; jaundice; the production of methæmoglobin by chemical poisons, such as potassic chlorate, or by excessive heat; or the presence of blood in the uriniferous tubes).

3. *Irritative* (from the presence of uric-acid infarctions or hæmatoidin infarctions, of purpuric or other interstitial hæmorrhages, or of microbes and toxines in the numerous eruptive and infectious maladies and in enteritis).